Mycotic aneurysm of the ascending aorta following CABG

M Prech, S Grajek, A Ciesliński, M Jemielity

Abstract
Mycotic aneurysm of the thoracic aorta is a rare and life threatening condition. Two patients are presented (both male, aged 66 and 59 years) in whom coronary artery bypass surgery was complicated by the development of a mycotic aneurysm. Fever preceded the radiological and echocardiographic signs of the aneurysm by at least several months in both cases. Blood cultures were negative for one patient and the source of Corynebacterium sp infection in the other was not determined for several months. Both patients died before surgery could correct the aneurysm.

Case 1
In May 1995, a 66 year old man was admitted to the cardiology department with fever and cachexia; he had a history of hypertension and inferior myocardial infarction. In June 1994, he had had a multivessel revascularisation for triple vessel coronary disease, and in December 1994 he was admitted with a recurrence of fever, which had appeared for the first time two months earlier. Laboratory studies (white blood cell count (WBC) 6.3 × 10⁹/l, erythrocyte sedimentation rate (ESR) 43 mm in the first hour, negative blood cultures), radiological examination (normal chest roentgenogram), echocardiography (aortic root diameter 3.0 cm in the transthoracic assessment), endoscopy, scintigraphy, and other tests did not reveal the origin of the fever. After two weeks of antibiotics the patient was discharged afebrile and did well for three months. However, in March 1995 he was admitted with septicemia. Corynebacterium sp (susceptible to imipenem, vancomycin, and ciprofloxacin) was isolated from blood cultures. The source of infection

Figure 1 (A) Magnetic resonance image: a large aneurysm of the ascending aorta (AA); arrows point to the mouth of the aneurysm. (B) Aneurysm of the ascending aorta (mouth of aneurysm between arrows) at necropsy. AO, aorta; PA, pulmonary artery; RA, right atrium; LV, left ventricle.
was not clear and after two months of treatment with vancomycin the surgical wires (the supposed cause of the fever) were removed from the sternum and he was moved to the cardiology department.

On admission, he was febrile and cachectic, and complaining of chest pain. Laboratory investigations showed anaemia, normal WBC count (but with a shift to the left), ESR 12 mm in the first hour, deteriorated parameters of renal function, and negative blood cultures. *Pseudomonas aeruginosa* was isolated from urine. The dimensions of the heart chambers and the aorta were normal on transthoracic echocardiography, whereas chest radiography showed a widened mediastinum. An aneurysm of the ascending aorta (presumably mycotic) was diagnosed by transoesophageal echocardiography. Magnetic resonance imaging confirmed a large (6.0 cm wide, 7.2 cm long) aneurysm of the ascending aorta (fig 1A). Despite high doses of antibiotics the patient developed septic shock and died before surgery. An aneurysm of the ascending aorta encompassing ostia of the venous grafts was found at necropsy (fig 1B). Histological examination of the aneurysm wall tissue (covered with bacterial vegetation) revealed complete destruction of elastin fibres (fig 2A and B) and lymphocytic inflammatory infiltrations (fig 2C).

**Case 2**

In May 1996 a 59 year old man was admitted to the department of cardiology because of syncope. His medical history included hyperthyreosis, hypertension, inferior myocardial infarction, and diabetes mellitus. He underwent vein bypass of the left anterior descending artery and right posterior descending artery on 11 September 1995. The same day he was reoperated owing to haemorrhage caused by the rupture of the ascending aorta. He was febrile in March 1996 and the following month endophthalmitis was diagnosed.

On admission he was blind, afebrile, and cachectic. He complained of diarrhoea. Laboratory investigations revealed anaemia, WBC $28 \times 10^9/l$, ESR 76 mm in the first hour, and raised serum creatinine. An initial blood culture was negative. Chest radiography showed a widened mediastinum (fig 3). An aneurysm of the ascending aorta (up to 9 cm diameter) was observed in transthoracic echocardiography. The patient died suddenly before any further investigations or surgery could be performed. Postmortem examination showed a large aneurysm of the ascending aorta. Histological examination, similar to case 1, showed complete destruction of elastin fibres and large inflammatory infiltrations of the aorta wall consisting of lymphocytes, monocytes, and proliferated capillaries (fig 2D).

**Discussion**

Mycotic aneurysm (also called infected false aneurysm or acute bacterial aortitis) is a localised abnormal dilatation of the arterial wall that
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precise diagnosis is difficult, especially in the early stages because of lack of specific symptoms. In most reported cases (including both of our patients) fever, the most common symptom of the disease, preceded radiological and echocardiographic signs of aneurysm formation by (at least) several months.

We conclude that in febrile patients who have undergone any invasive procedure of the cardiovascular system, meticulous investigation including transthoracic echocardiography or magnetic resonance imaging might help to diagnose the cause of the fever. Once a mycotic aneurysm is diagnosed, surgical treatment with in situ insertion of an aortic homograft or synthetic graft should be done without delay. Pharmacological treatment alone is ineffective and will not save the patient's life.

Extramural damage, an inherent part of surgery, and infiltration of the aortic wall with bacteria and inflammatory cells triggers the process of aneurysm formation. Different inflammatory infiltrations were observed in both presented cases (fig 2C and D). Virulent bacteria—staphylococci and salmonellae most often isolated from the blood of patients diagnosed with mycotic aneurysms—usually cause an intense inflammatory reaction. Once started, however, further dilatation of the aortic wall might be independent of the presence of bacteria. In some cases (including case 2) less virulent organisms or no organisms are isolated.

Matrix metalloproteinases localised to inflammatory cells (mainly monocyte derived macrophages) and smooth muscle cells cause elastin and collagen breakdown (fig 2A and B) and consequent dilatation of the aortic wall. Freestone et al suggested that the rate of aneurysm expansion may reflect the intensity of inflammation within the aortic wall.

There are probably other factors that affect the process of aneurysm formation: the extent of the intimal damage—the more severe the destruction of the aortic wall, the easier it will be for bacteria and inflammatory cells to infiltrate; immunological disorders which enhance the development of mycotic aneurysms especially in transplant patients and pharmacological treatment—antibiotics and anti-inflammatory drugs might slow the process of aneurysm enlargement. Unfortunately, a conservative approach to mycotic aneurysms cannot prevent further dilatation of the aortic wall as the damaged elastin fibres are not resynthesised in the adult aorta.

Mycotic aneurysm of the aorta should be suspected in patients with fever of unknown origin who have undergone any invasive procedure of the cardiovascular system. However,
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